The neuron as a temporal electroacoustic medium.

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The human brain is one of the most complex and intriguing scientific topics. The most stablished theory of neuronal communication is a pure electrical model based on the propagation of intracell cationic charges along the neurons. Here we propose a complementary model based on two properties of brain communication: A) The large Coulomb repulsion within the intracell Na+ cation pulse at the Action Potential (AP) induces a deformation of the neuron membrane which travels as an acoustic signal, i.e.: the intracell ion pulse makes both the electric and acoustic signals be strongly correlated. B) As brain communication is stablished through a periodic train of AP pulses it induces a time periodic modulation of the acoustic parameters. In this framework we propose envisaging the neuron as a temporal electro-acoustic medium. The temporal varying media framework could help understanding brain conundrums such as propagation routes involved in the neuronal plasticity in the consolidation of the memory, as well as on the generation of the signals associated to the brain field theory.

The brain is the most complex system of the human body. Brain activity is distributed over an intricate network of neurons (approximately 10^{11}), where the transmission and management of information is carried out through neural connections (about 10^3 per neuron) called synapses. One of the most important subjects of inquiry in neurology concerns understanding how the brain can receive, manage, and record experiences and sensations, as well as later evoke memories and emotions. The extraordinary process of integration is performed through the communication between different areas of the brain in a gigantic network some authors called it as the brain web[1] or the connectome.[2,3]

The more accepted model for brain communication is a pure electrical model developed by Hodgkin and Huxley in 1952[4]. However, first in 1980 Tasaki and collaborators[5] and later other groups around the world[6,7,8,9], reported on the swelling of nerve fibers associated to the Action Potential (AP) pulse, pointing out to an acoustic wave propagating together with the electrical pulse. From a schematic vision, the propagation of the acoustic signal in a neuron would be like the sound propagation along a cylindric shaped long membrane filled with the neuron fluid (essentially an ionic solution) immersed in practically the same neuron fluid. Due to the different acoustic parameters of the wave components of the AP pulse the pulse propagation is usually distorted and at a certain distance its integrity is compromised. In 2009 Heimburg and Jackson[10] proposed an acoustic wave model based on soliton transmission, addressing this problem. Here, the non-lineal properties of the medium, attributed to a phase change of the lipidic membrane, compensate the dispersion. In this way the shape of the pulse is preserved and the soliton is propagated. Later, Schneider et al. provided experimental support to solitary solutions[11,12]. Although the soliton model is a very interesting proposal it does not include the role of the intracell ion cloud.

Here we present an electroacoustic model of neuronal communication based on the physicochemical properties associated to the action potential (AP) pulse. Although this model is naïve and it must be experimentally tested, we believe it presents all ingredients to be considered as a framework of discussion which might provide some hints for unveiling the complex nature of brain processes. The model is based on two closely related key points of brain communication: A) The AP induces a large transient concentration of Na^+ cations producing a strong electrostatic repulsion within the intracell Na^+ ion cloud. Therefore, the electrostatic repulsion induces a mechanical deformation of the neuron membrane which, in turn, due to flexoelectricity[13], produces a change of the membrane potential. However, and more important, the electrostatic repulsion within the Na^+ ion cloud induce a dramatic change of the intracell fluid stiffness

responsible of the acoustic wave propagation, i.e.: the electrostatic repulsion produces a temporal modulation of the acoustic parameters associated to the AP pulse. Finally, this model might give arguments for understanding the heating-cooling cycle appearing in the AP pulse[14]. B) Brain communication is stablished through a train of pulses. Assuming that, (i) the medium is perturbed and modulated by these acoustic pulses and considering that (ii) these signals are periodic or quasiperiodic in time, the neuron could be envisaged as a temporal electro-acoustic medium.

In the literature, time varying medium has postulated as a framework for obtaining a plethora of wave phenomena such as dynamic changes of wave propagation,[15] propagation through the border (edge states),[16] wave generation,[17] resonant behavior[18] and amplification.[18,19,20] Our aim concerns showing the neuron might be a temporal electro-acoustic medium candidate which may help understanding the complex phenomena of brain communication.

Results and discussion

1. Electro-acoustic coupling

The AP pulse produces huge concentration of intracell Na^+ cations. Because of the electrostatic repulsion within the Na^+ ion cloud, a measurable neuron membrane deformation has been reported.[5, 21,22] Let us analyze it from the physicochemical point of view. The AP is triggered by the flux of a large amount Na+ ions into the intracellular neuron fluid of the order of $[Na^+] \approx 165$ mM.[23] It produces a time dependent ion concentration pulse that propagates along the neuron which corresponds to a huge Na^+ ion density value around $\sigma \approx 10^{20}$ cm^{-3}. To understand how large this figure is let us compare it to the density of electrons appearing in well-known materials used in Electronics and Material Science[24]. The $[Na^+]$ ion concentration within the action potential pulse is ten times larger than the impurity concentration of a doped degenerate semiconductor like silicon and only two orders of magnitude smaller than the electron concentration of a very good metal like gold

Figure 1. un-myelinated neuron model.

($\sigma_{(Au)}$=5.9 x 10^{22} cm^{-3}).[24] A very simple calculation shows that at the AP pulse ($[Na^+] \approx 150$ mM) the mean distance between intracell Na ions is around 2 nanometers (nm). This produces a strong Coulomb repulsion[24] which triggers a mechanical deformation of the neuron membrane.[5,21] As a result, it induces a transversal acoustic wave along the neuron membrane. Also, as the neuron is immersed in the extracellular brain fluid, solely the intracellular Na^+ ion cloud is what makes stiff the neuron fluid. Therefore, the electrostatic repulsion of the Na^+ ion cloud should be included in the calculation of the stiffness value, a key parameter which controls the velocity of the AP acoustic wave. As depicted in figure 1 the un-myelinated neuron model is built up by a cylindrical membrane filled with the neuron fluid (essentially water plus the Na^+ ion cloud) both immersed in the extracellular fluid, essentially water. As the neuron membrane is surrounded by water, and this medium present zero shear elasticity, we assume that the relevant factors which govern the transversal acoustic wave propagation are both the compressibility parameters of the Na^+ ion cloud, κ_{ion}, and the surface compressibility of the lipid membrane κ_A.

Cell membrane	[Na+] mM	σ (cm^{-3})	a (nm)	κ_{ion} (Pa)$^{-1}$	c_{max} (ms^{-1})
depolarized	165	9.9·10^{19}	2.2	5.41·10^{-6}	13.6
half-depolarized	83	5.0·10^{19}	2.7	1.6·10^{-5}	8.6
polarized	15	9.0·10^{18}	4.8	9.2·10^{-5}	2.7

Table 1. Na$^+$ ion concentration (mM and cm^{-3}), nearest neighbor Na$^+$ ion distance in nanometers, compressibility parameter, κ_{ion}, and phase velocity, c, for three stages of the AP.

Therefore, the total compressibility, κ, of the neuron can be written as,

$$\frac{1}{\kappa} = \frac{1}{\kappa_A} + \frac{1}{\kappa_{ion}} \quad (1)$$

The sound velocity, c, in a fluid is then given by the following expression[25]

$$c = \sqrt{B/\rho}; \quad B = \frac{1}{\kappa} \quad (2)$$

being B and ρ, the bulk modulus and the density values of the fluid medium, respectively. Table 1 shows the compressibility values κ_{ion} and the inter-ionic distance, a, of the Na^+ cloud at the maximum value of the AP (full depolarization) as well as half-depolarization obtained through the expression (S5). See the Supporting Information for a detailed development of κ_{ion}. As in both cases, the compressibility value of the neuron surface membrane takes very large values,[10,26] $(\kappa_A(37^\circ C) = 5,4 \ (Pa)^{-1})$ we conclude that $\kappa_A \gg \kappa_{ion}$, and $\kappa \approx \kappa_{ion}$. As $\kappa_A \gg \kappa_{ion}$ then $\kappa \approx \kappa_{ion}$.

It is important emphasizing this is a very simple model since it does not consider other properties of the neuron membrane as its diameter or its surface viscosity. It is important pointing out that κ_{ion} is not a constant parameter, but it is a time dependent parameter which value changes about one order of magnitude during the AP pulse (see point 2, Time varying electroacoustic medium). As the acoustic properties of the neuron change with time we assume that the mean value of phase velocity corresponds to the half-depolarization of the AP pulse (see Table 1); i.e.: $c = 8.6$ m/s, of the same order of magnitude of those reported for un-myelinated neurons.[27]

This model also might give response to the large velocity values of the AP pulse in the case of myelinated neurons. Myelin covering layer also produces a mechanical hardening of the neuron surface this probably decreasing the surface compressibility factor κ_A, but not as small as the compressibility due to the electrostatic repulsion of the ion cloud. The concentration of Na^+ channels, N, at the nodes of Ranvier increases enormously the intracell Na^+ ion flux producing a dramatic increase of the electrostatic repulsion. It is known that the density of Na^+ channels in myelinated axons, at the nodes of Ranvier, is around 26 times larger than those of unmyelinated axons.[28,29,30] If we assume all Na^+ ion channels provide the same ion flux, the ratio between the pulse velocity of myelinated and unmyelinated axons would be (see Supp. Information)

$$c_{myel}/c_{unmyel} = \left(N_{myel}/N_{unmyel}\right)^{2/3} = 8.8$$

i.e.: the AP pulse in myelinated axons should travel around 9 times faster than that of unmyelinated axons.

Table 2 shows the calculated values of the compressibility parameter κ and the mean values of the phase velocity for unmyelinated and myelinated neurons. For more details on the calculation of the stiffness coefficient see the Supporting Information.

Axon	κ (Pa)$^{-1}$	c(ms^{-1})
Unmyelinated (depolarized)	$1.55 \cdot 10^{-5}$	8.6
Myelinated (depolarized)	$2.0 \cdot 10^{-7}$	75.2

Table 2. Compressibility parameter κ, see equation (S5), and phase velocity, c, obtained from equation (2).

We can see the large differences in the value of the compressibility parameter κ for myelinated and unmyelinated axons at the depolarized stages of the AP. We must emphasize this is a first and very simple approach which gives a rough estimation of the pulse velocity of the same order of magnitude as those experimentally observed[27].

The above model can also explain the origin of the heating/cooling cycle associated to the AP signal[14]. Several interpretations to the observed heating/cooling cycle have been published[14,31,32]. Of especial interest is the proposal given by Abbott et al. in 1958[31], and later revisited by Chandler et al.[33] This model which was able to partially account for the heat production/absorption is based on the free energy charge/discharge cycle of the neuron membrane capacitor. Our model goes on

the line of the Abbott proposal, but it suggests including the compression/expansion cycle of the Na^+ ion cloud as an additional mechanism for the heat emission/absorption mechanism; i.e.: the intracell Na^+ ion accumulation produced by the AP pulse can be envisaged as a gas cloud submitted to a compression/expansion process which might result on a heating/cooling Carnot cycle like it happens in a refrigerator machine.

2. Time varying electroacoustic medium.

Brain communication is stablished through a train of AP pulses which customarily are periodic or quasiperiodic in time. As the AP pulse strongly modifies the compressibility parameter of the intracell fluid, the pulse train produces a quasiperiodic modulation of the acoustic properties of the neuronal fluid. So, the sound velocity c in the medium and the compressibility κ parameters are time periodic functions i.e.:

$$c\ (t) = c\ (t+T),\ \kappa\ (t) = \kappa\ (t+T),\ (3)$$

being T the firing rate of the AP pulse.

The propagation of waves in media whose parameters are periodic in time has been of great interest for the Metamaterials community. They are called as Temporal Photonic Crystals[17,18,34,35,36] or Photonic Time Crystals[19,20] in the case of Electromagnetic Waves, and Temporal Phononic Crystals[37,38] in the case of acoustic waves. However, this research topic was for many years of great interest to the electronic engineering community under the name of Time Varying Dielectric Media.[39,40,41]

Figure 2. Frequency ω vs k-vector dispersion relation in normalized units for the relative compressibility parameter $\kappa'_M = 5.8 \times 10^{-2}$

One of the fingerprints of a time varying media is the appearance of certain wave-vector regions, called as gaps where the wave is not allowed to be propagated through it. These gaps also appear in media whose either optical or acoustic properties are periodic in the space,[42,43] and they are called as Photonic Crystals[44,45] or Sonic Crystals[46] respectively. However here gaps appear in the frequency region.

As a neuron can be modelled as a narrow and long cylinder of radius R (see Figure 1), we assume the acoustic signal is propagating along a one-dimensional (1D) system. Here, the membrane swelling is produced by the sound pressure $p_R(z,t)$ across the radius and it propagates along the cylindrical axis of the neuron (z direction).

Then, by analogy to the Electromagnetic (EM) case the sound wave equation can be written as,[17,25]

$$\frac{\partial^2}{\partial z^2} p_R(z,t) - \frac{\partial^2}{\partial t^2}\left[p_R(z,t)\Big/\left(c(z,t)\right)^2\right] = 0\ (4)$$

$$c^2 = 1\big/\rho\kappa = c_0^2\kappa_0\big/\kappa\ (5)$$

c_0 and κ_0 being the sound velocity and the compressibility of the intracellular fluid at the polarized stage, respectively. Here the fluid density, ρ, is a constant parameter (we can roughly assume the intracell fluid is essentially water).

After an algebraic development like that of Temporal Photonic Crystals[18] (see de Supp. Information) we obtain the following canonical equation:

$$\sum_m\left[\sum_{p-m}\alpha_p(\omega)\kappa'_{p-m}(\omega) - k^2c_0^2\delta_{pm}\right]q_m(\omega) = 0\ (6)$$

$$p, m\ = 0, \pm1, \pm2, \pm3\ ....$$

$$\alpha_p(\omega) = (\omega - p\Omega)^2$$

being δ_{pm}, $\kappa'_{p-m}(\omega)$ and $q_m(\omega)$ the Kronecker delta tensor, the Fourier components of the relative compressibility, and the sound wave pressure amplitude respectively.

Equation (6) can be regarded as the interaction of a transverse acoustic signal of frequency ω propagating in the intracell medium (the axon) with a compressibility, κ (t), periodic in time originated by the periodic train of pulses of frequency $\Omega = 2\pi/T$.

After introducing the Fourier components of the relative compressibility parameter (see the Supp. Information) into equation (6), we get the frequency vs k-vector value of the acoustic modes in the neuron. Figure 2 shows the solution of equation (6); i.e.: it shows the dispersion relation $\omega(k)$ of the propagating neuronal acoustic modes (figure 2). This result is like the dispersion obtained in a transmission line of Reyes-Ayona and Halevi,[47] but with some specific properties different to that of Photonic Time Crystal. In the following paragraph we explain and discuss the obtained results.

The blue lines of Figure 2 represent the available acoustic wave parameters (ω vs k-wavevector) or solutions of the acoustic wave travelling through the neuron. These solutions show the typical periodic pattern in the vertical axes (frequency). In the literature where usually, the medium is modulated in time by a sinusoidal wave, the period of this pattern corresponds to its single frequency. By contrast, the modulation in this study is created by pulses, each one composed by multiple waves (see Fig S1b). In contrast to the usual single gap of the literature, here multiple gaps (gray regions) have been obtained. This is due to both, the presence of multiple wave components of the AP pulse (Fig. S1b) as well as the strong modulation of the compressibility parameter (Fig. S1a).

Next, we will discuss some physiological consequences of considering the neuron as time-varying medium. The presence of the time-varying electroacoustic medium represents a dynamic and malleable behavior of the media. In particular, the peculiarities of the train of pulses would modify or determine the wave propagation, including effects of reflection suppression[48] and/or amplification[18]. i.e.: the temporal crystal configuration could dynamically influence different responses at axon branch points, such as reflection, conduction block and full conduction[49,50], as well as amplification effects.[18,51] It might produce cellular modifications as was first visioned by Donald Hebb[52] and later demonstrated by Eric Kandel[53] and many others concerning neural networks in the process of the consolidation of the memory. [54,55,56,57]

In line with the brain field theory, the multidirectional combined response of time varying electro-acoustic media could give rise to collective effects. Consequently, frequency generation, long range resonances and amplification[17,18] could be revealed. This might give theoretical support to the resonant modes appearing in the brain field theory for mapping brain activity.[58,59]

2. Conclusions

In conclusion, here we present an electroacoustic model of neuronal communication based on two properties of the AP pulse.

A) The electrostatic repulsion of the intracell Na^+ ion cloud at the AP pulse induces a mechanical deformation of the neuron membrane, this triggering an acoustic wave which propagates together with an electric pulse, i.e.: both the electric and the acoustic signal are strongly correlated.

B) The periodic distribution of the train of pulses induces a time periodic modulation of the acoustic parameters which may envisage the brain as a time periodic electro-acoustic medium. The temporal varying media framework could open numerous possibilities of wave phenomena to consider. This provides a theoretical background for effects such as the dynamic modification of the propagation phenomena as well as wave generation and amplification. This framework could contribute to the progress of knowledge of the routes involved in the neuronal plasticity and in the consolidation of the memory, as well as the generation of the signals associated to the brain field theory.

3. Limitations and particularities of the model

1 – Although in the first part of the work we have calculated de values of the phase velocity, a more realistic model for calculating the group velocity, considering the membrane properties, would be necessary.

2 – Role of other ions Ca+, K+, Cl- etc. Role of neurotransmitters and other intracell species in the wave propagation phenomena.

3 – The train of pulses are not infinite in time but they are composed by a finite number of pulses. Therefore, broadening of the peaks as well as windowing effects are expected. Therefore, a more realistic scheme considering these peculiarities could match better a bounded time scheme.

4 – In the temporal crystal formulation, the spatial terms are not included. Therefore, the time varying compressibility concerns a bounded area (such as the cell body) or a certain discrete part of the transmission line.[47] The inclusion of the spatial modulation could add more properties of wave transmission as it has been reported in many theoretical studies[60,61,62] such as non-reciprocity propagation of the electro-acoustic wave. Non reciprocity means that the AP pulse propagates in the forward direction. In any case, a hypothetical additive effect of counter propagating waves (such as back reflections in axon branch points), could create spatial regions of strong effects of time varying media.

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